

CASE OF THE WEEK

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CLINICAL PICTURE

CLINICAL PICTURE:

33 Years old male patient presented clinically with neurogenic claudication of the cauda equina. Clinical examination revealed depression of the left ankle reflex. (To inspect the patient's full radiological study, click on the attachment icon (The paper clip icon in the left pane) of the acrobat reader then double click on the attached file) ([Click here](#) to download the attached file)

RADIOLOGICAL FINDINGS

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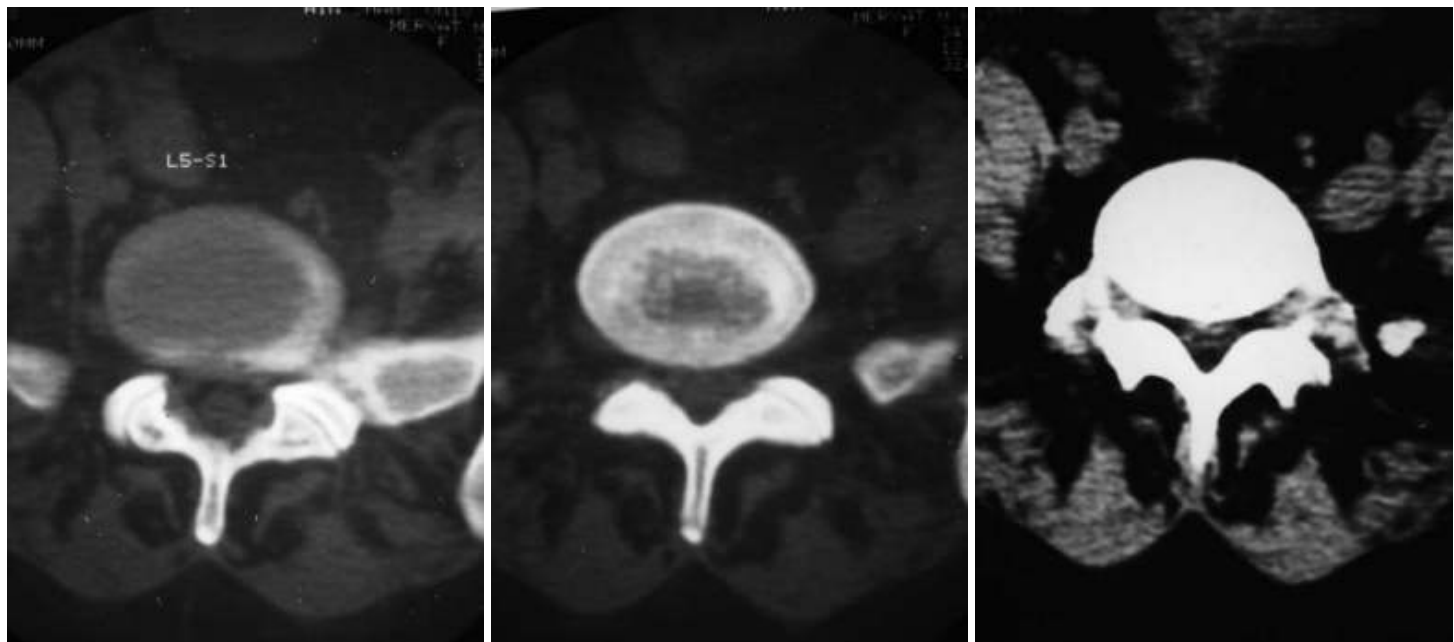


Figure 1. Plain CT scan lumbar spine showing bony stenosis of the lumbar canal (antero-posterior diameter is 11.5 CM). Notice mild annular bulging, articular fact pathology (asymmetric orientation, medial osteophytosis, and reduction of the synovial joint space). The lumbar canal is trefoil in shape.

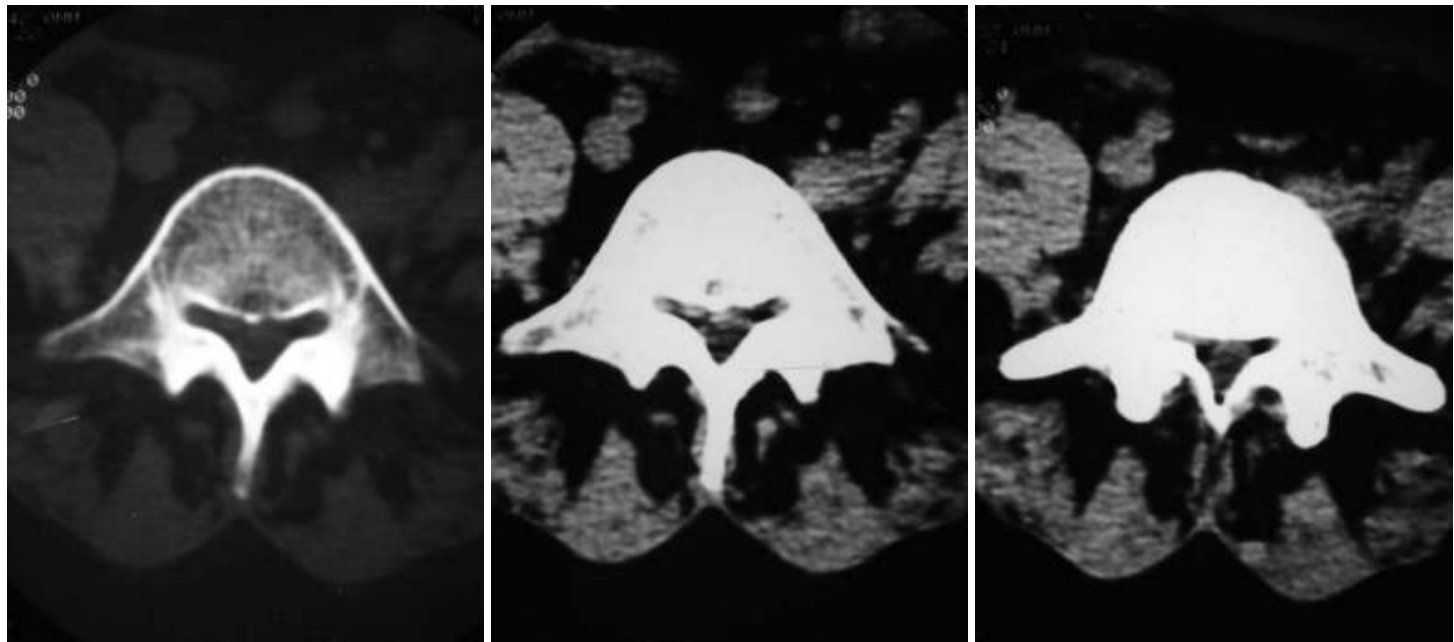


Figure 2. Plain CT scan lumbar spine showing bony stenosis of the lumbar canal (antero-posterior diameter is 11.5 CM). Notice articular fact pathology (asymmetric orientation, medial osteophytosis, and reduction of the synovial joint space and some facet hypertrophy). The lumbar canal is trefoil in shape.

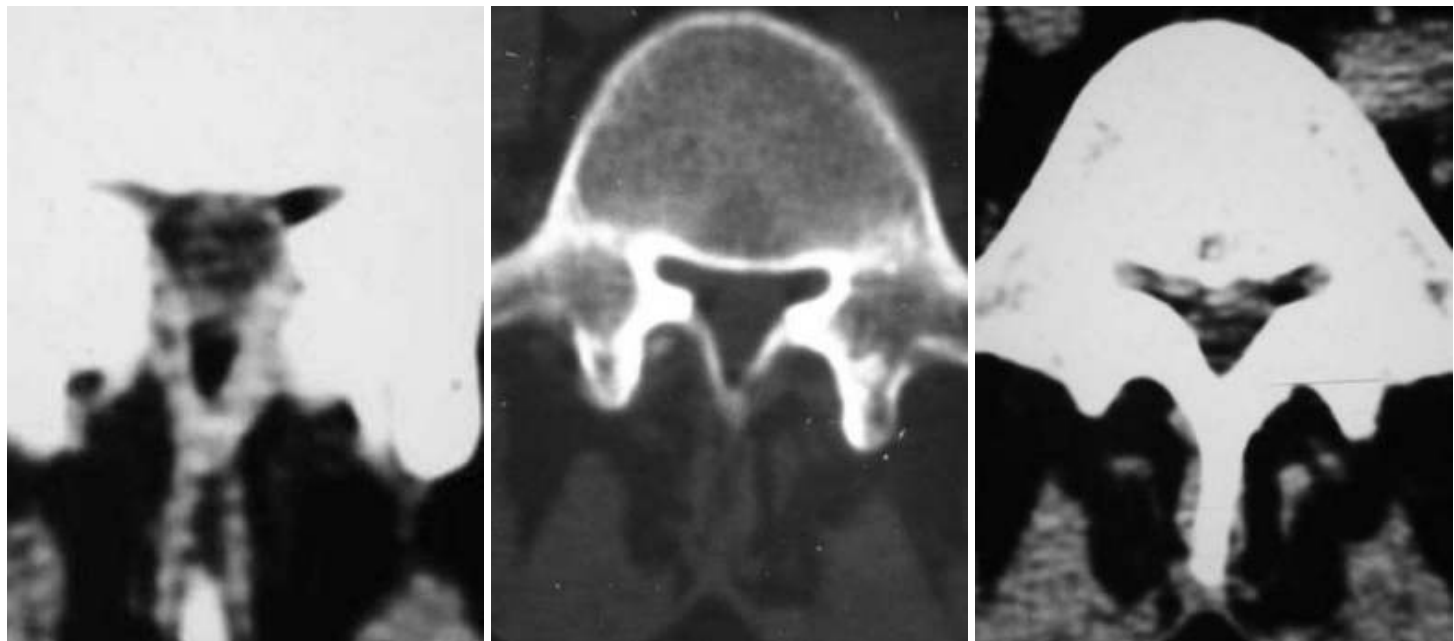


Figure 3. Plain CT scan lumbar spine showing bony stenosis of the lumbar canal (antero-posterior diameter is 11.5 CM). Notice ligamentum flavum hypertrophy. The lumbar canal is trefoil in shape.

Spinal stenosis describes a syndrome characterized by neurogenic claudication with exertion. Spinal canal stenosis can be congenital or acquired.

- **Congenital spinal stenosis**

Congenital spinal stenosis constitutes 10% to 15% of stenosis cases. It is generally idiopathic in nature, consisting of developmental hypoplasia of the posterior arch with short thick pedicles that narrow the AP dimension of the central canal. The central canal may have a trefoil shape in cross section. Less common causes of congenital central canal stenosis include achondroplasia, Morquio's syndrome, and other bony dysplasias. Isolated facet hypertrophic arthropathy or PEH (which alone can cause radiculopathy) can results in trefoil-shaped narrowing. The trefoil

appearance characteristic of central canal stenosis might also be due to a combination of zygapophysial joint and ligamentum flavum hypertrophy.

- **Degenerative central canal stenosis**

Degenerative central canal stenosis constitutes most cases of clinical spinal stenosis, including mild cases of idiopathic congenital central canal narrowing that (with the addition of degenerative change) become clinically significant. Encroachment on the central canal may occur anteriorly by disc bulge, herniation, or end plate hypertrophy. Posterolaterally, osteoarthritis involving the facet joints or associated synovial cysts may exert mass effect on the central canal. True synovial cysts communicate with the facet joint and are lined by synovial tissue; the indistinguishable ganglion cyst does not communicate with the joint. Ninety percent of synovial cysts occur in the lumbar region; 75% of lumbar synovial cysts occur at the L4-5 level, likely related to its considerable flexion and extension motion [2]. They generally are seen in the elderly (aged 50–70). Synovial cysts may exhibit hemorrhage, peripheral calcification, or an enhancing inflammatory reaction. Hence, synovial cysts typically are heterogeneous in their imaging characteristics.

Absolute measurements of the spinal, in general, is not very useful. The morphologic appearance of the lumbar spine, the trefoil shape of the short pedicle stenosis, the complete loss of epidural fat, and the relative overgrowth of the facets and/or the ligamentum flavum are much more useful in assessing stenosis.

DIAGNOSIS:

DIAGNOSIS: CONSTITUTIONAL LUMBAR CANAL STENOSIS

DISCUSSION

DISCUSSION:

Background: Lumbar spinal stenosis (LSS) implies spinal canal narrowing with possible subsequent neural compression. LSS is classified by anatomy or etiology. Anatomic subclassifications include central canal and lateral recess stenosis.

- Central canal stenosis, commonly occurring at an intervertebral disc level, defines midline sagittal spinal canal diameter narrowing that may elicit neurogenic claudication (NC) or pain in the buttock, thigh, or leg. Such stenosis results from ligamentum flavum hypertrophy, inferior articulating process (IAP), facet hypertrophy of the cephalad vertebra, vertebral body osteophytosis, and herniated nucleus pulposus (HNP).
- Lateral recess stenosis (ie, lateral gutter stenosis, subarticular stenosis, subpedicular stenosis, foraminal canal stenosis, intervertebral foramen stenosis) is defined as narrowing (less than 3-4 mm) between the facet superior articulating process (SAP) and posterior vertebral margin. Such narrowing may impinge the nerve root and subsequently elicit radicular pain. This lateral region is compartmentalized into entrance zone, mid zone, exit zone, and far-out stenosis.
 - The entrance zone lies medial to the pedicle and SAP, and, consequently, arises from facet joint SAP hypertrophy. Other causes include developmentally short pedicle and facet joint morphology, as well as osteophytosis and HNP anterior to the nerve root. The lumbar nerve root compressed below SAP retains the same segmental number as the involved vertebral level (eg, L5 nerve root is impinged by L5 SAP).
 - The mid zone extends from the medial to the lateral pedicle edge. Mid-zone stenosis arises from osteophytosis under the pars interarticularis and bursal or fibrocartilaginous hypertrophy at a spondylolytic defect.
 - Exit-zone stenosis involves an area surrounding the foramen and arises from facet joint hypertrophy and subluxation, as well as superior disc margin osteophytosis. Such stenosis may impinge the exiting spinal nerve.
 - Far-out (extracanalicular) stenosis entails compression lateral to the exit zone. Such compression occurs with far lateral vertebral body endplate osteophytosis and when the sacral ala and L5 transverse process

impinge on the L5 spinal nerve.

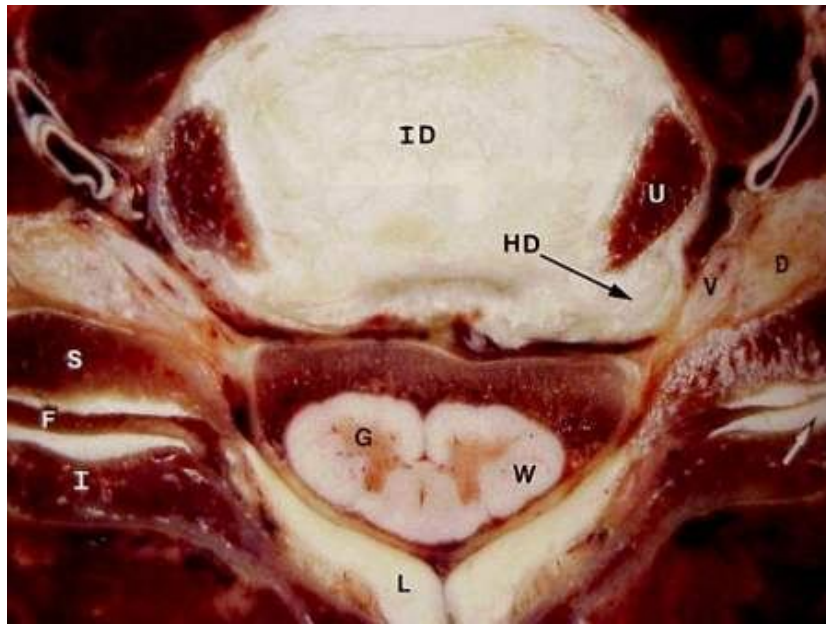


Figure 1. Normal lumbar disc anatomy. Notice that it is difficult to differentiate between the nucleus pulposus and annulus fibrosus

ID = Intervertebral disc

S= Superior articular facet

I = Inferior articular facet

L = Ligamentum flavum

G = Gray matter

W= white matter

F= Facet joint

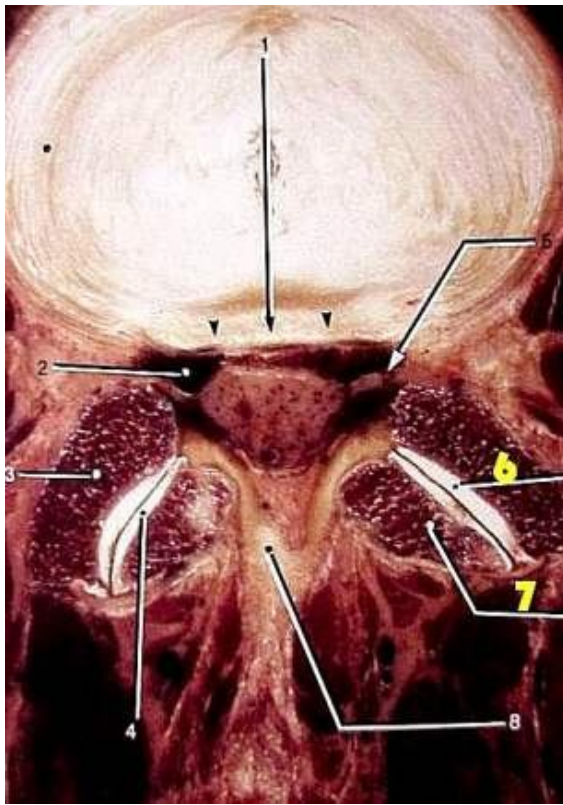


Figure 2. Normal lumbar disc anatomy. Notice that it is difficult to differentiate between the nucleus pulposus and annulus fibrosus

1. The intervertebral disc
2. Epidural fat
3. Superior articular facet
4. Inferior articular facet
5. Spinal roots
6. Superior articular joint
7. Inferior articular facet
8. Tegmentum flavum

Amundsen and colleagues found concomitant lateral recess stenosis in all cases of central canal stenosis; consequently, in his study, pure central stenosis without lateral stenosis failed to exist.

Parenthetically, Keim and colleagues mention the following simplistic LSS anatomical classification scheme:

- Lateral, secondary to SAP hypertrophy
- Medial, secondary to IAP hypertrophy
- Central, due to hypertrophic spurring, bony projection, or ligamentum flavum/laminar thickening
- Fleur de lis (clover leaf), from laminar thickening with subsequent posterolateral bulging

LSS arises from the following primary and secondary etiologies:

- **Primary stenosis** encompasses congenital malformations and developmental flaws. Congenital malformations include incomplete vertebral arch closure (spinal dysraphism), segmentation failure, achondroplasia, and osteopetrosis. Developmental flaws include early vertebral arch ossification, shortened pedicles, thoracolumbar kyphosis, apical vertebral wedging, anterior vertebral beaking (Morquio syndrome), and osseous exostosis.
- **Secondary (acquired) stenosis** arises from degenerative changes, iatrogenic causes, systemic processes, and trauma. Degenerative changes include central canal and lateral recess stenosis from posterior disc protrusion, zygapophyseal joint and ligamentum flavum hypertrophy, and spondylolisthesis. Iatrogenic changes result following surgical procedures such as laminectomy, fusion, and discectomy. Systemic processes that may be involved in secondary stenosis include Paget disease, fluorosis, acromegaly, neoplasm, and ankylosing spondylitis.

Pathophysiology: Disc desiccation and degenerative disc disease (DDD) with resulting loss of disc height may induce segmental instability. Such instability incites vertebral body and facet joint hypertrophy. Cephalad vertebral body IAP hypertrophy promotes central spinal canal stenosis. Further canal volume loss results from HNP, ligamentum flavum hypertrophy, and disc space narrowing.

Alternatively, the caudal vertebral body SAP contributes to lateral recess and foraminal stenosis. Indeed, facet hypertrophy between L4 and L5 vertebrae may impinge the L4 nerve root in the foramen and the L5 proximal nerve root sheath in the lateral recess.

Jenis and An eloquently describe foraminal stenosis pathoanatomy, characterized by disc desiccation and DDD, which narrows disc height, permitting the caudad SAP to sublux anterosuperiorly. Such subluxation decreases foraminal space. Continued subluxation with resulting biomechanical disruption provokes osteophytosis and ligamentum flavum hypertrophy, further compromising foraminal volume. Anteroposterior (transverse) stenosis ultimately results from narrow disc height and hypertrophy anterior to the facet; specifically, the SAP and posterior vertebral body transversely trap the nerve root. Furthermore, in vertical (craniocaudal) stenosis, posterolateral vertebral endplate osteophytes and a lateral HNP may impinge the spinal nerve against the superior pedicle.

Dynamic foraminal stenosis implies intermittent lumbar extension-provoked nerve root impingement from HNP, osteophytosis, and vertebral body slippage. Such dynamic stenosis with associated intermittent position-dependent symptoms may not manifest on imaging studies, thereby confounding diagnosis. Other factors promoting development of LSS include shortened gestational age, and synovial facet joint cysts with resulting radicular compression. Adult degenerative scoliosis, secondary to DDD-induced instability with subsequent vertebral rotation and asymmetric disc space narrowing, promotes facet hypertrophy and subluxation in the curve concavity. Degenerative spondylolisthesis, when combined with facet hypertrophy, causes both central canal and lateral recess stenosis.

The pathophysiology of claudication is not completely understood, however it is believed that it is caused by venous pooling / congestion induced by impairment of venous drainage by stenosis at the root level and will only occur if stenosis is present at a minimum of two adjacent vertebral levels.

- **Degenerative lumbar spinal stenosis** results from pathological degeneration of the facet joints, disc herniation, hypertrophy and buckling of the ligamentum flavum, and spondylolisthesis. Degenerative changes in the three-joint complex (the intervertebral disc and two facet joints) explain the fluctuation in symptoms with alterations in posture, load, and duration of load. Lumbar intervertebral disc degeneration represents a cascade of events involving disc herniation, bulging of the disc and ligamentum flavum into the canal, and resulting chronic facet arthrosis, sclerosis, and osteophytic growth. Hypertrophy of the ligamentum flavum is also an important element in the development of spinal stenosis. Lumbar spinal encroachment induces ligamentum flavum hypertrophy, which further aggravates stenosis. Disease of the nerve roots and cord, however, does not typically result directly from compression of the nerves. Rather, the resulting stenosis causes decreased flow of cerebrospinal fluid, which represents approximately 60% of the nutritional supply to the cauda equina, and increased venous pressure. In such a scenario, any concurrent spinal deformities may critically compromise the nerve roots and cord, as well as exacerbate neurological symptoms of lumbar stenosis.
- **Spondylolisthesis** can be caused by congenital, developmental, traumatic, neoplastic, or degenerative conditions. In degenerative spondylolisthesis, the most common type observed with lumbar stenosis,

anterior/posterior displacement of a VB results from facet joint erosion and attenuation of the muscular, capsular, and ligamentous structures. Fourfold more common in females than in males, degenerative spondylolisthesis occurs most frequently at the L4-5 and L5-S1 levels. Disc degeneration causes spondylolisthesis with resulting segmental hypermobility, compounded by arthritis in sagittal facet joints. These facet joints, which support up to one third of the spinal static compression load and axial loads, lose resistance to shear forces in the sagittal plane. The resulting degenerative anterior vertebral subluxation, which is usually no greater than 30% of the VB width, and vertebral instability can contribute to the neurogenic claudication of spinal stenosis. Isthmic spondylolisthesis results from bilateral defects in the pars interarticularis; a widening of the central canal occurs as the posterior arch of the cephalad vertebrae remains in place while the VB displaces anteriorly. Although this represents a direct widening of the spinal canal, callus formation around the pars defect can induce overall stenosis of the canal. Spondylolisthesis is often associated with back pain, which significantly influences the surgeon's selection of treatment methods.

- Scoliosis may complicate lumbar stenosis. Most cases can be classified as degenerative, idiopathic, or postsurgical. Idiopathic scoliosis is present when degenerative changes occur in cases of preexisting idiopathic scoliosis. Degenerative scoliosis occurs in previously nonscoliotic spines in which disc and facet joint disease cause coronal deformity. Finally, postsurgical scoliosis may result from surgery-induced compromise of posterior elements or the intervertebral disc. With a prevalence of 3% in the general population, idiopathic scoliosis may contribute significantly to the presentation of lumbar stenosis. Degenerative scoliosis is characterized by loss of lumbar lordosis, vertebral slippage (either lateral or anterior) at one or more levels, relatively short curves, and moderate amplitudes of scoliotic curvature. Asymmetrical vertebral collapse and listhesis contribute to the compromise of nerve roots, and may cause back pain and coronal decompensation. Thus, scoliosis complicates both the presenting symptoms as well as the treatment of lumbar stenosis.

Box 1. The role of venous congestion in symptom formation in spinal canal stenosis

- The pathophysiology of claudication is not completely understood, however it is believed that it is caused by venous pooling / congestion induced by impairment of venous drainage by stenosis at the root level and will only occur if stenosis is present at a minimum of two adjacent vertebral levels.
- Proposed mechanisms for development of NC include cauda equina microvascular ischemia, venous congestion, axonal injury, and intraneural fibrosis. Ooi and colleagues myeloscopically observed ambulation-provoked cauda equina blood vessel dilation with subsequent circulatory stagnation in LSS patients with NC. They propose that ambulation dilates the epidural venous plexus, which, amidst narrow spinal canal diameter, increases epidural and intrathecal pressure. Such elevation of pressure ultimately compresses the cauda equina, compromises its microcirculation, and causes pain.
- Canal stenosis causes venous congestion of the cauda equina at any spinal segment. At rest this is not a problem, but with the activity of walking, the arterioles in the congested segment probably fail to vasodilate, causing nerve root dysfunction.

Frequency:

- In the US: LSS remains the leading preoperative diagnosis for adults older than 65 years who undergo spine surgery. The cost of more than 30,000 LSS surgeries performed in 1994 exceeds 1 billion dollars.

The incidence of lateral nerve entrapment is reportedly 8-11%. Some studies implicate lateral recess stenosis as the pain generator for 60% of patients with symptomatology of failed back surgery syndrome.

Incidence of foraminal stenosis increases in lower lumbar levels because of increased dorsal root ganglion (DRG) diameter with resulting decreased foramen (ie, nerve root area ratio). Jenis and An cite commonly involved roots as L5 (75%), L4 (15%), L3 (5.3%), and L2 (4%). The lower lumbar levels maintain greater obliquity of nerve root passage, as well as higher incidence of spondylosis and DDD, further predisposing patients to L4 and L5 nerve root impingement.

Mortality/Morbidity: In their review of lumbar spinal stenosis, Fritz and colleagues cite several studies suggesting that many patients show symptomatic and functional improvement or remain unchanged over time. For example, they mention Porter and colleagues' study in which 90% of 169 untreated patients with suspected lateral recess

stenosis improved symptomatically after 2 years. Additionally, they report Johnson and colleagues' 4-year study of 32 patients treated conservatively for moderate stenosis, of whom only 16% worsened clinically and 30% reported diminished walking tolerance.

Race: No known correlation exists between incidence of LSS and race.

Sex: Patients typically are male.

Age: Patients with LSS due to degenerative causes generally are aged at least 50 years; however, LSS may be present at earlier ages in cases of congenital malformations.

Spinal canal stenosis: Pathology, anatomy and pathogenesis

• Anatomy and pathology

The spinal cord in adults ends at the upper border of the L1 vertebral body and continues as multiple nerve roots, the cauda equina, that descend to their specific neural foramina, providing exit from the lumbosacral spinal canal. The spinal canal ranges from 15 to 23 mm in its anteroposterior diameter and is a triangular space bounded anteriorly by the dorsal surfaces of the bodies of the lumbar vertebrae and the disk spaces (covered by the posterior longitudinal ligament), medially by the pedicles that extend from the lateral margin of the vertebral body, posteriorly by the laminae of the vertebral arch and their covering, the ligamentum flavum; and the facet joints that are part of the posterior elements of each vertebral body. The vertebral bodies are connected to each other by the disks anteriorly and two facet or zygoapophyseal joints posteriorly. The disks are composed of a tough outer connective tissue annulus fibrosis and a soft, jelly-like center, the nucleus pulposus.

There are two major categories of lumbar spinal stenosis: congenital and acquired. The major contributors to narrowed lumbar canals on a congenital basis are short pedicles, thickened lamina and facets, and excessive scoliotic or lordotic curves. Patients who have these congenital anatomic changes have a small safety factor for the emergence of clinically significant lumbar spinal stenosis, which may be precipitated by further canal narrowing from later life-onset superimposed degenerative joint changes. Defects in cellular metabolism leading to retardation of cartilaginous growth and irregular intracartilagenous bone formation lead to congenital spinal stenosis in achondroplastic dwarfism, where there is significant narrowing of the spinal canal in all its dimensions, especially in the upper lumbar regions because of shortened pedicles, hypertrophied zygapophyseal joints, and thickened laminae.

The majority of cases of lumbar spinal stenosis, however, are acquired, and stem from degenerative or arthritic changes that affect the three-joint complex between lumbar vertebrae: the two zygoapophyseal (facet) joints posteriorly and the adjoining intervertebral disk anteriorly. The degenerative process begins most often with the disk and affects the articular processes secondarily. Initially, there is desiccation of the disk, narrowing of the disk space, rents or fissures in the annulus, disk bulging, and frank herniation of nucleus pulposus. This soon is followed by hypertrophic degenerative changes of the facets (osteophyte formation) and thickening of the ligamentum flavum. This results in central narrowing, so that the anteroposterior diameter is attenuated (typically to less than 12 mm), with compression of the cauda equina, and lateral narrowing (at the recesses), with root compression at the entrance of the intervertebral foramen. Spondylolysis (a defect in the pedicles, the pars interarticularis—congenital or acquired) may lead to spondylolisthesis, the anterior displacement of one vertebra relative to the one beneath it, further narrowing an already stenotic lumbar canal.

The spine is the site affected second most commonly in Paget's disease, predisposing patients to spinal stenosis, occurring in one third of those who have spinal involvement. Pagetic spinal stenosis is brought about by bone remodeling of lumbar vertebrae, with bone expansion in all directions: posteriorly from the vertebral bodies, anterioromedially from the vertebral lamina, and medially from the pedicles. This leads to hypertrophic facet arthropathy and resulting spinal stenosis. Some cases of neurologic deterioration do not result from direct compression of neural elements, rather from spinal ischemia resulting from diversion of blood flow through remodeled hypervascular pagetic bone (referred to as the arterial steal phenomenon).

• Pathogenesis

Three explanations are advanced to explain the phenomenon of neurogenic claudication, the cardinal manifestation of lumbar spinal stenosis. They are designated the postural, the ischemic, and the venous stasis

(stagnant hypoxia) theories. The postural theory suggests that symptoms are explained by transient compression of the cauda equina (leading to sensory and motor axon dysfunction) by degenerated intervertebral disks and thickened ligamenta flava, when the lumbar spine is extended and lordosis is accentuated, either at rest or in the erect posture. In the ischemic theory, it is proposed that the metabolic demand of the cauda equina cannot be met during activity (eg, walking), that blood flow needs of the lumbosacral nerve roots are not met by the local vasculature that is compromised by lumbar spinal stenosis. Porter suggested the venous stasis theory: that the underlying mechanism of neurogenic claudication is inadequate oxygenation or the accumulation of metabolites in the cauda equina. He presented evidence from a porcine model that venous pooling of one or more nerve roots of the cauda equina between two levels of low pressure stenosis transitions to venous engorgement during exercise (walking), that in turn tends to prevent the expected arteriolar vasodilation response to activity, leading to nerve conduction failure with resulting symptoms of tiredness, weakness, and discomfort in the legs when walking.

CLINICAL PICTURE

History: LSS classically presents as bilateral neurogenic claudication (NC). Unilateral radicular symptoms may result from severe foraminal or lateral recess stenosis. Patients, typically aged more than 50 years, report insidious-onset NC manifesting as intermittent, crampy, diffuse radiating thigh or leg pain with associated paresthesias. Indeed, leg pain affects 90% of patients with LSS.

NC pain is exacerbated by standing erect and downhill ambulation and is alleviated with lying supine more than prone, sitting, squatting, and lumbar flexion. Getty and colleagues documented 80% pain diminution with sitting and 75% with forward bending. Lumbar spinal canal and lateral recess cross-sectional area increases with spinal flexion and decreases with extension. Furthermore, cross-sectional area is reduced 9% with extension in the normal spine and 67% with severe stenosis. The Penning rule of progressive narrowing implies that the more narrowed the canal by stenosis, the more it narrows with spinal extension. Schonstrom and colleagues have shown that spinal compressive loading from weight bearing reduces spinal canal dimensions.

NC, unlike vascular claudication, is not exacerbated with biking, uphill ambulation, and lumbar flexion and is not alleviated with standing. LSS patients compensate for symptoms by flexing forward, slowing their gait, leaning onto objects (eg, over a shopping cart) and limiting distance of ambulation. Unfortunately, such compensatory measures, particularly in elderly osteoporotic females, promote disease progression and vertebral fracture. Pain radiates downward in NC and, in contrast, upward in vascular claudication. Hall and colleagues note the presence of radiculopathy in 6% and NC in 94% of LSS patients.

Proposed mechanisms for development of NC include cauda equina microvascular ischemia, venous congestion, axonal injury, and intraneural fibrosis. Ooi and colleagues myeloscopically observed ambulation-provoked cauda equina blood vessel dilation with subsequent circulatory stagnation in LSS patients with NC. They propose that ambulation dilates the epidural venous plexus, which, amidst narrow spinal canal diameter, increases epidural and intrathecal pressure. Such elevation of pressure ultimately compresses the cauda equina, compromises its microcirculation, and causes pain.

Canal stenosis causes venous congestion of the cauda equina at any spinal segment. At rest this is not a problem, but with the activity of walking, the arterioles in the congested segment probably fail to vasodilate, causing nerve root dysfunction.

Another pain generator may be the dorsal root ganglion (DRG), which contains pain-mediating neuropeptides, such as substance P, that possibly increase with mechanical compression. The DRG varies spatially within the lumbosacral spine, with L4 and L5 DRG in an intraforaminal position and S1 DRG located intraspinally. Such foraminal placement may predispose to stenotic compression with subsequent radicular symptomology.

Lastly, severe radiologic stenosis in otherwise asymptomatic individuals suggests inflammation, not just mechanical nerve root compression. Specific inflammation generators may include HNP, ligamentum flavum, and facet joint capsule.

Katz and colleagues report that the historical findings most strongly associated with LSS include advanced age, severe lower extremity pain, and absence of pain when the patient is in a flexed position. Fritz and colleagues contend that the most important elements involve the postural nature of the patient's pain, stating that absence of pain or improvement of symptoms when seated assists in ruling in LSS. Conversely, LSS cannot be ruled out when sitting is the most comfortable position for the patient and standing/walking is the least comfortable.

Physical: Physical examination findings frequently are normal in patients with LSS. Nevertheless, review of the literature suggests diminished lumbar extension appears most consistently, varies less, and constitutes the most significant finding in LSS. Other positive findings include loss of lumbar lordosis and forward-flexed gait. Radiculopathy may be noted with motor, sensory, and/or reflex abnormalities. Asymmetric muscle stretch reflexes and focal myotomal weakness with atrophy occur more with lateral recess than central canal stenosis. Some report objective neurologic deficits in approximately 50% of LSS cases. Provocative maneuvers include pain reproduction with ambulation and prone lumbar hyperextension. Pain alleviation occurs with stationary biking and lumbar flexion.

Negative findings in the physical examination include skin color, turgor, and temperature; normal distal lower extremity pulses; absence of arterial bruits; and unremarkable dural tension signs. Lumbar segment mobilization often fails to reproduce pain, and palpation locates no trigger points.

Katz and colleagues report physical examination findings most strongly associated with LSS include wide-based gait, abnormal Romberg test, thigh pain following 30 seconds of lumbar extension, and neuromuscular abnormalities; however, Fritz and colleagues state physical examination findings do not seem helpful in determining the presence or absence of LSS.

Johnsson and colleagues' single study of the natural course of LSS reports unchanged symptoms in 70% of patients, improvement in 15%, and worsening in 15% after a 49-month observation period. Walking capacity improved in 37% of patients, remained unchanged in 33%, and worsened in 30%.

WORKUP

Lab Studies:

- Lab studies are not necessary to support the diagnosis of LSS.

Imaging Studies:

- Plain radiograph (x-ray)
 - Nonspecific plain radiographic findings possibly implicating LSS include the following:
 - Disc space narrowing
 - Facet hypertrophy and arthrosis
 - Spondylosis
 - Degenerative scoliosis and spondylolisthesis
 - Osteochondrosis
 - Transitional segmentation
 - Spinous process settling
 - Shortened interpedicular distance
 - Interpedicular distance, considered subnormal if less than 18 mm, commonly increases from upper to lower lumbar segments.
 - Some sources define pure absolute central canal stenosis as a mid-sagittal canal diameter of less than or equal to 10 mm, pure relative at 10-12 mm, and mixed as a combination thereof. Mid-sagittal canal diameter less than 15 mm and transverse diameter less than 20 mm usually are considered abnormal.
 - Posterior disc height of 4 mm or less and foraminal height of 15 mm or less may suggest foraminal

stenosis; nevertheless, clinical correlation is required. No convincing correlation has been found between clinical symptoms and radiologic findings in a study of 100 symptomatic patients with LSS. Similarly, no correlation has been shown between physical function and radiologic findings.

- CT scan

- CT scan provides excellent central canal, lateral recess, and neuroforaminal visualization. Additionally, CT scan offers contrasts between intervertebral disc, ligamentum flavum, and thecal sac. Unfortunately, CT scan, like MRI, yields a high false-positive rate (35.4% when correlated with surgically proven LSS).
- Parasagittal reconstructed CT scan findings suggesting stenosis include posterolateral vertebral body or facet osteophytosis extending into the foramen.

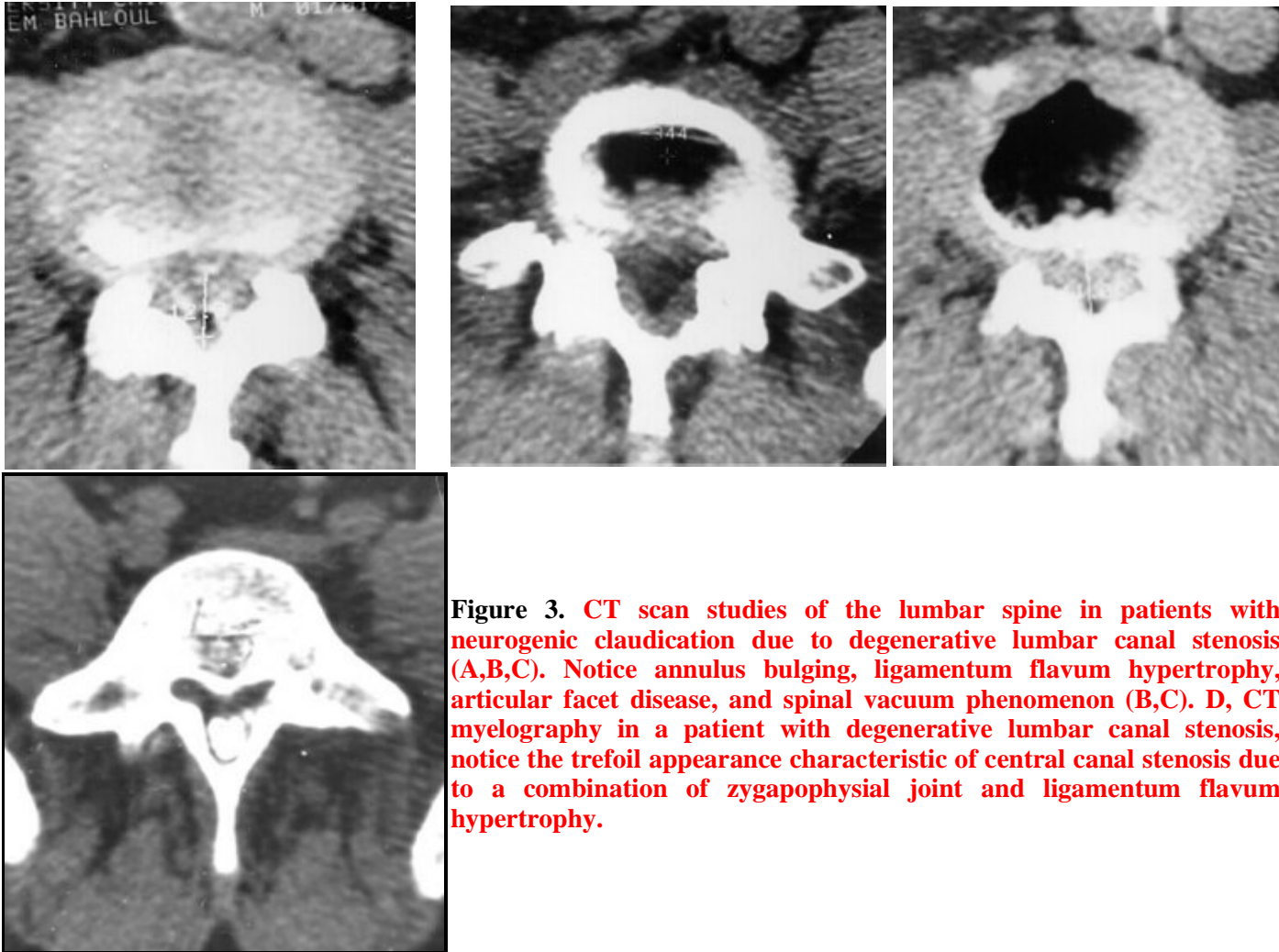


Figure 3. CT scan studies of the lumbar spine in patients with neurogenic claudication due to degenerative lumbar canal stenosis (A,B,C). Notice annulus bulging, ligamentum flavum hypertrophy, articular facet disease, and spinal vacuum phenomenon (B,C). D, CT myelography in a patient with degenerative lumbar canal stenosis, notice the trefoil appearance characteristic of central canal stenosis due to a combination of zygapophysial joint and ligamentum flavum hypertrophy.

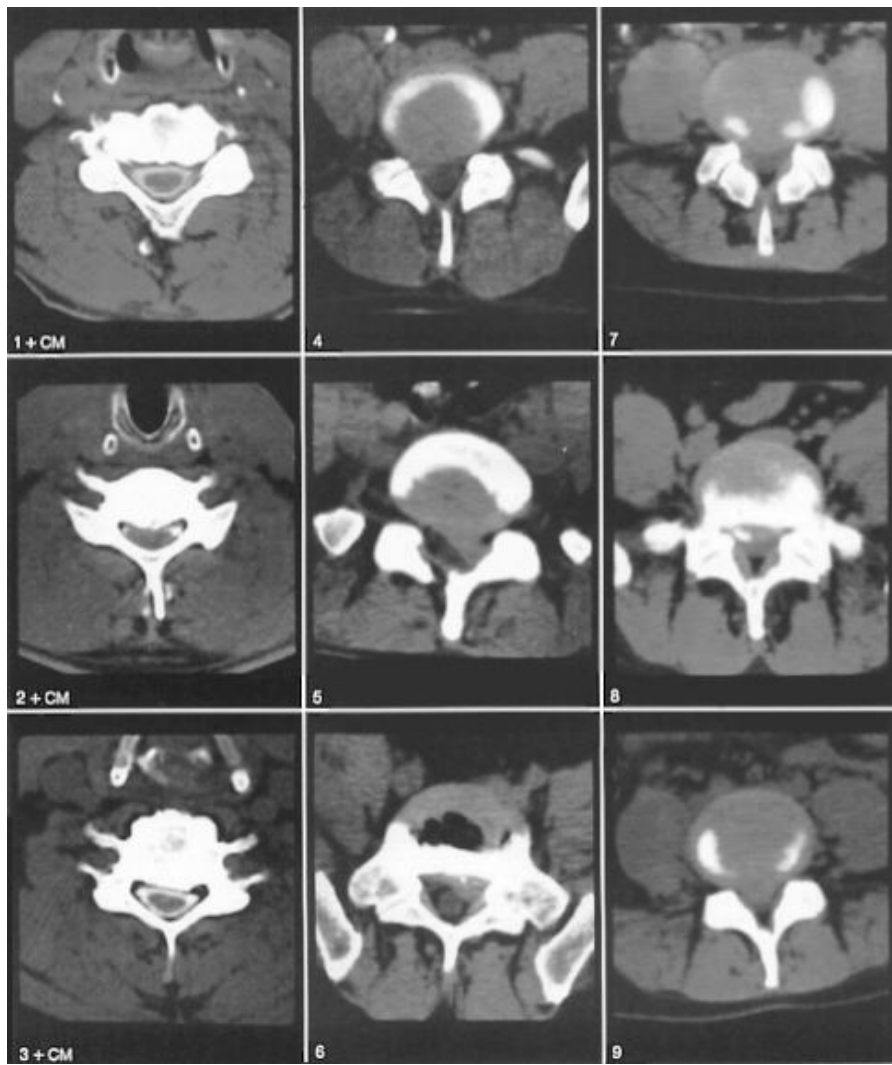


Figure 4. CT myelography of the lumbar spine showing spondylitic changes in the form of annulus bulge, ligamentum flavum hypertrophy, articular facet degenerative changes, spinal vacuum phenomenon and disc herniation collectively inducing lumbar canal stenosis in a patient presented with neurogenic claudication of the cauda equina

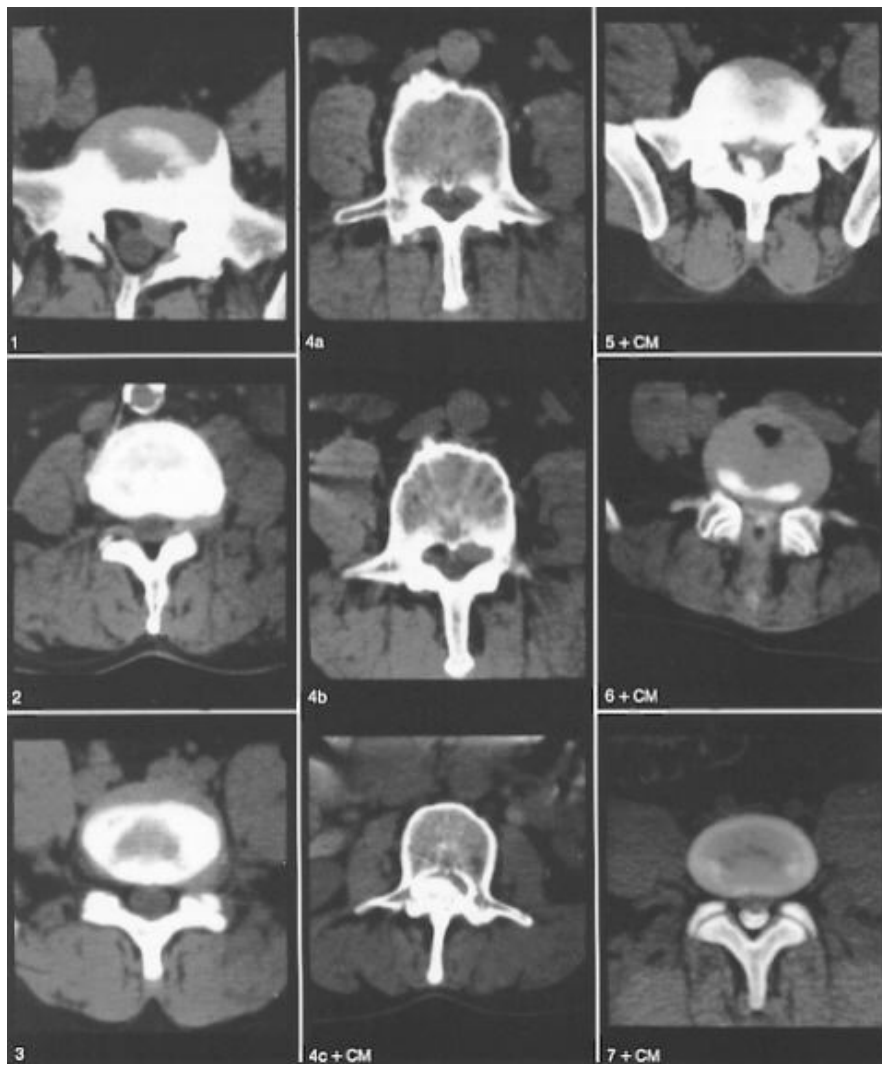


Figure 5. CT myelography of the lumbar spine showing spondylitic changes in the form of ligamentum flavum hypertrophy, articular facet degenerative changes, spinal vacuum phenomenon and disc herniation collectively inducing lumbar canal stenosis in a patient presented with neurogenic claudication of the cauda equina



Figure 6. CT myelography of the lumbar spine showing spondylitic changes in the form of annulus bulge, ligamentum flavum hypertrophy, articular fact degenerative changes and spinal vacuum phenomenon collectively inducing lumbar canal stenosis in a patient presented with neurogenic claudication of the cauda equina

- MRI

- MRI remains the imaging modality of choice for LSS. Fritz and colleagues maintain that MRI effectively rules LSS in or out anatomically.
 - Advantages include nonionizing radiation and superior multiplanar soft tissue visualization without osseous artifact. A trefoil-shaped central spinal canal may provoke more symptoms than a round or oval canal by depressing the lateral recess.
 - Sagittal T1-imaged adipose tissue outlines neuroforaminal nerve root segments and dorsal root ganglia. Therefore, parasagittal MRI findings suggesting foraminal stenosis include paucity of T1-weighted perineural adipose tissue surrounding the nerve root and diminished foraminal size. Unfortunately, MRI abnormalities have been documented in 20% of asymptomatic subjects.

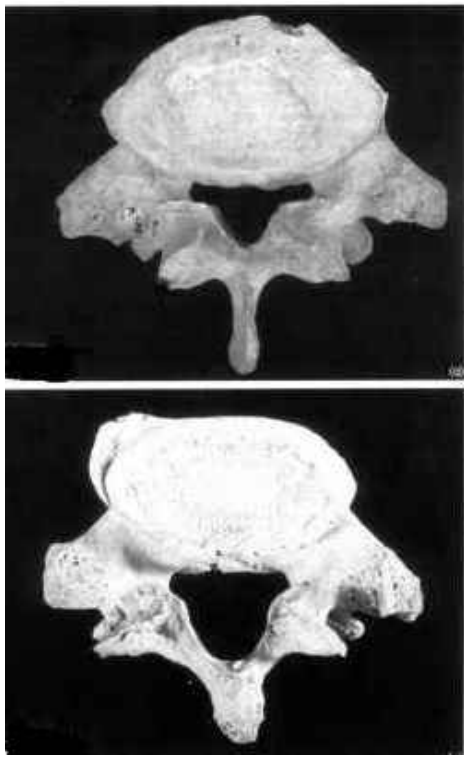
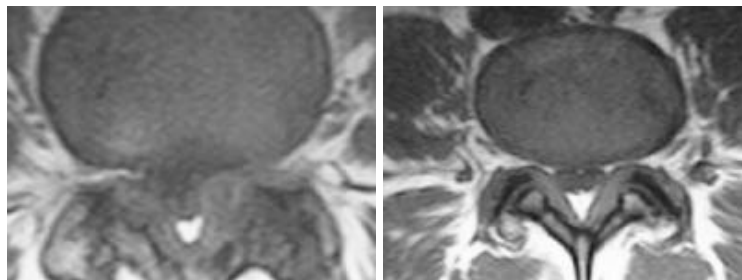


Figure 7. Compares two 5th lumbar vertebrae. One vertebra has a large canal (B) whilst another has a shallow trefoil canal (A).

- **Myelography**

- This test effectively documents central canal stenosis and remains superior in evaluating lumbar disc herniation. Predictive value of myelography versus CT scan has been reported as 83% versus 72%, respectively, for lumbar disc herniation, and 93% versus 89% for LSS. Furthermore, myelography images the entire lumbar spinal canal, and enhances stenotic segments due to hyperextension during imaging; however, it may miss lateral stenosis and HNP because the dural sac terminates at the lateral mid zone, preventing contrast spread to the distal nerve root sheath.
- Myelography is less sensitive and specific than CT scan or MRI.
- Procedural complications include spinal headache, seizure, allergic reaction, and nausea.



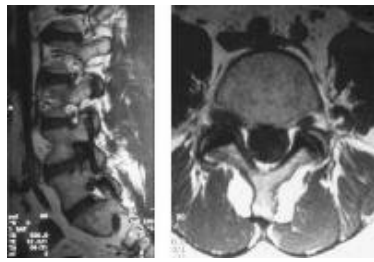


Figure 8. A, Lateral T2-weighted MRI demonstrating narrowing of the central spinal fluid signal (L4-L5), suggesting central canal stenosis. B, Axial T2 MRI image (L4-L5) of the same patient as in (A) confirming central canal stenosis. C, Trefoil appearance characteristic of central canal stenosis due to a combination of zygapophysial joint and ligamentum flavum hypertrophy. D, Lateral and axial MRI demonstrating right L4 lateral recess stenosis secondary to combination of far lateral disc protrusion and zygapophysial joint hypertrophy

Other Tests:

- **Electrodiagnosis (EDX), including needle electromyography (EMG), nerve conduction studies (NCS), and somatosensory evoked potentials (SSEP), evaluates nerve root and peripheral nerve function.**
 - **Needle EMG diagnoses lumbosacral radiculopathy by detecting increased insertional activity, spontaneous potentials (eg, positive waves, fibrillations, fasciculations, chronic repetitive discharges), and decreased motor unit recruitment in paraspinal and lower extremity muscles innervated by the same nerve root.**
 - **Limitations include inability to evaluate sensory and upper motor neurons.**
 - **Multisegmental muscle innervation may cause false negative results by preserving motor unit function despite nerve root compromise. Such innervation may elicit multilevel abnormalities in severe LSS.**
 - **Johnsson and colleagues have correlated myelographic LSS severity with multisegmental EMG abnormality.**
 - **NCS differentiates LSS from other confounding neuropathic conditions such as lumbosacral plexopathy, generalized peripheral neuropathy, and mononeuropathy (eg, peroneal neuropathy at the fibular head, tarsal tunnel syndrome).**
 - **Canal stenosis may compress the cauda equina with resulting polyradicular insults. Such multiple lumbosacral radiculopathies involve lower lumbosacral (especially S1) nerve roots, are often bilateral and asymmetric, and frequently may manifest NCS abnormalities. Such abnormalities include decreased or unelicitable posterior tibial and peroneal compound motor action potentials (CMAPs) reflecting axon loss, and unobtainable H reflexes signifying bilateral S1 compression. Sensory nerve action potentials (SNAPs) remain unaffected (unless impingement occurs distal to the dorsal root ganglion), but may not be detectable in older persons.**
 - **Wilbourn and Aminoff advocate measuring peroneal CMAP amplitude from tibialis anterior and M-wave amplitude during H-reflex testing to gauge the extent of L5 and S1 acute denervation, respectively.**
 - **Overall, Wilbourn and Aminoff report variable EDX findings, including multiple, bilateral lumbosacral radiculopathies in 50% of LSS patients, with prominent chronic motor unit action potential (MUAP) changes, and fibrillations solely in distal musculature. The remaining 50% of patients demonstrate varied abnormalities, with some manifesting 2 radiculopathies commonly as a single radicular insult in each lower extremity, either symmetrically (eg, bilateral L5) or asymmetrically (eg, left S1 and right L5). Other patients display isolated L5 or S1 radiculopathy. Limited nondiagnostic findings may be elicited, including bilaterally absent H reflexes with normal lower extremity needle EMG and sural SNAPs, as well as fibrillations in a single S1-innervated limb muscle. Lastly, many patients demonstrate normal EDX tests.**

increased soft tissue flexibility in LSS patients.

Medical Issues/Complications: In rare cases, central canal stenosis may provoke cauda equina syndrome with associated saddle anesthesia, bladder and/or bowel dysfunction and altered muscle reflexes. Additionally, patients with lateral recess stenosis-induced radiculopathy may manifest significant lower limb weakness or numbness. Lastly, intractable axial, radicular, or neurogenic claudication pain may result.

Surgical Intervention: LSS remains one of the most common conditions leading to lumbar spine surgery in adults aged 65 years and older. Increasing rates of LSS surgery among the Medicare population has been shown to be due possibly to imaging techniques that enable physicians to diagnose LSS more frequently. Other contributing factors may include improved surgical techniques that might allow patients previously managed conservatively to undergo surgery, as well as a philosophy that LSS surgery prevents future morbidity.

- Widely agreed upon indications for LSS surgery do not exist. Typically, patients undergo elective surgery to improve walking tolerance and disabling leg and back pain. Preoperatively, such disability infrequently is measured in objective quantitative terms. Some suggest preoperative treadmill testing to facilitate objective selection of potential surgical candidates. Surgical emergencies (eg, cauda equina syndrome, rapid neurologic deterioration) rarely arise.
 - Surgical techniques include standard wide laminectomy and decompression, which first removes lamina and ligamentum flavum from the lateral borders of one lateral recess to the other and then decompresses entrapped nerve roots.
 - Foraminal enlargement surgery is used to address refractory foraminal stenosis-induced radicular pain. Other surgical decompressions include the following:
 - Laminotomy
 - Medial facetectomy
 - Medial or lateral foraminotomy.
 - Midline interlaminar approaches are used to address concurrent central and foraminal stenosis.
 - The Wiltse approach with foraminotomy is used for isolated foraminal stenosis by providing the following:
 - Widening the longissimus-multifidus muscle interval
 - Removing the lateral pars interarticularis and facet joint
 - Exposing the nerve root with subsequent decompression
 - In addition to decompression and foraminal enlargement, some patients with segmental instability from facet joint removal and pain secondary to DDD may require fusion.
 - Fusion stabilizes the intervertebral segment while maintaining lordosis and foraminal size.
 - Additional options include arthrodesis and instrumentation.
- Surgical outcomes for patients with LSS vary.
 - Surgical outcome literature is difficult to assess due to observer bias, inadequate outcome data categorization, vaguely defined outcome measures, and study design.
 - Reports show widely varied outcomes (26-100% success and 31% dissatisfaction at 4.6 years), due to disparate research methodologies.

- **Conservative versus surgical treatment for LSS remains controversial due to wide variations in outcome study type and quality.**
 - **Johnsson and colleagues document improvement in 60% of surgically treated patients with 25% worsened, compared with improvement in 30% of conservatively treated patients and no change in 60%.**
 - **Atlas and colleagues tracked 67 conservatively treated and 81 surgically treated patients over 12 months; surgically treated patients reported greater improvement in pain relief than those treated conservatively.**
 - **Treatment outcome predictors do not exist; specifically, severe spinal degenerative changes do not necessarily correlate with an unfavorable prognosis or mandate surgery.**
 - **Simotas and colleagues cite that 12 of 49 patients treated conservatively with incorporation of analgesics, physical therapy, and epidural steroid injection, reported sustained improvement. Conservative and surgical treatments have not been subjected to rigorous well-designed study.**

Other Treatment (injection, manipulation, etc.): Epidural steroid injection (ESI) provides aggressive-conservative treatment for LSS patients who demonstrate limited response to oral medication, physical therapy, and other noninvasive measures. Corticosteroids may inhibit edema formation from microvascular injury sustained by mechanically compressed nerve roots. Furthermore, corticosteroids inhibit inflammation by impairing leukocyte function, stabilizing lysosomal membranes, and reducing phospholipase A2 activity. Lastly, corticosteroids may block nociceptive transmission in C fibers. When using oral steroids (in rapid tapering fashion), remember that possible side effects may include fluid retention, skin flushing, and shakiness. Local anesthetic may be combined with corticosteroids to provide immediate pain relief and diagnostic feedback on the proximity of the injectate to the putative pain generator.

- **Caudal ESI**

- **Caudal ESI entails needle placement through the sacral hiatus into the sacral epidural space.**
- **Advantages include ease of performance and low risk of dural puncture.**
- **Disadvantages include large injectate volumes (6-10 mL) necessary to ensure adequate medication spread to more cephalad pathology (ie, above L4-L5). Furthermore, such large volumes potentially may dilute the effect of the corticosteroid.**

- **Interlaminar ESI**

- **Interlaminar ESI entails needle passage through the interlaminar space, with subsequent injection directly into the epidural space. Consequently, delivery of medication occurs closer to the affected spinal segmental level than in caudal ESI.**
- **Disadvantages include greater potential for dural puncture, and, like caudal ESI, limited spread of medication to the target site if a midline raphe or epidural scarring exists. Furthermore, interlaminar injection delivers medication to the posterior epidural space with possible limited ventral diffusion to nerve root impingement sites.**

- **Transforaminal ESI**

- **Transforaminal ESI facilitates precise deposit of higher steroid concentrations closer to the involved spinal segment, and, consequently, might prove more efficacious in reducing pain.**
- **Transforaminal ESI may be used for unilateral radicular pain provoked by lateral recess or foraminal stenosis.**
- **Bilateral transforaminal ESI also may be used to treat bilateral central stenosis-induced NC pain when**

imaging studies demonstrate limited posterior epidural space, thereby precluding safe interlaminar ESI. Otherwise, interlaminar ESI may be used to treat bilateral or multilevel NC or radicular pain.

- **Absolute contraindications to ESI include bleeding diathesis and anticoagulation therapy because of increased risk of epidural hematoma. Anticoagulation therapy (eg, warfarin, heparin) should be stopped a few days prior to injection. (Alternative methods of DVT prophylaxis, such as serial compression hose, should be instituted in the interim). In the case of patients taking coumadin, PT/INR should be drawn the day of the procedure. Aspirin and other nonsteroidal anti-inflammatory drugs (NSAIDs) should be discontinued before the procedure in accordance with their half-life and hematologic profile.**
 - **Other absolute contraindications include systemic infection, injectate allergy, and pregnancy (because of the teratogenicity of fluoroscopy). Relative contraindications include diabetes mellitus (DM) and congestive heart failure, given the hyperglycemic and fluid retention properties of corticosteroids, respectively. Other relative contraindications include adrenal dysfunction and hypothalamic-pituitary axis suppression.**
 - **Serious complications, although rare, include infection (eg, epidural or subdural abscess) and epidural hematoma. Dural puncture (in 5% of lumbar interlaminar ESIs and 0.6% of caudal injections) with possible subsequent subarachnoid anesthetic/corticosteroid deposition may provoke neurotoxicity, sympathetic blockade with hypotension, and/or spinal headache; however, contrast-enhanced fluoroscopic guidance minimizes the possibility of dural puncture and intravascular injection.**
 - **Therapeutic epidural steroid injection (ESI) techniques are performed ideally using fluoroscopic guidance and radiologic contrast dye enhancement to ensure delivery of injectate to the target site. Studies document misplacement of 40% of caudal and 30% of interlaminar injections performed without fluoroscopy, even by experienced injectionists.**
 - **Transient corticosteroid dose-related side effects include facial flushing, low-grade fever, insomnia, anxiety, agitation, hyperglycemia, and fluid retention. Steroids may suppress the hypothalamic-pituitary axis for 3 months following the injection. Lastly, vasovagal reaction, nerve root injury, injectate allergy, and temporary pain exacerbation can occur as well.**
 - **Recent studies assessing efficacy of fluoroscopically guided, contrast-enhanced ESI, even for HNP-induced radicular pain, appear promising, suggesting that a significant inflammatory component amenable to corticosteroid treatment may accompany HNP-nerve root pathology.**
 - **Studies of ESI for LSS treatment demonstrate mixed results due to varying injection and guidance techniques, patient populations, follow-up periods and protocols, ancillary treatments (eg, physical therapy, oral medication), and outcome measures. This lack of consistency limits the ability to assess ESI efficacy for LSS.**
 - **Some studies, nevertheless, suggest that, unlike HNP-provoked radicular pain, NC may be more mechanical or ischemic than inflammatory in nature. Consequently, corticosteroid anti-inflammatory properties may fail to provide designed long-term symptom relief. Studies report that 50% of patients with LSS or HNP-provoked radicular pain received temporary relief and that such results were close to those associated with the placebo effect.**
 - **Because of concomitant lateral recess stenosis from facet hypertrophy or lateral HNP, patients may fail transforaminal ESI therapy for HNP-induced radicular pain. ESI may do little to relieve chronic lateral recess stenosis-related radicular pain. Additionally, studies show patients with a preinjection duration of symptoms greater than 24 weeks may respond to ESI as favorably as those with symptoms of less than 24 weeks' duration. This finding, may suggest that chronic nerve compression could induce irreversible neurophysiologic change that ultimately renders the nerve root refractory to ESI.**
 - **Future studies require controlled design, contrast-enhanced fluoroscopic guidance, and objective validated outcome measures before definitive conclusions can be drawn regarding efficacy of ESI treatment of LSS.**
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SUMMARY

Spinal stenosis refers to narrowing of the spinal canal, lateral recesses or nerve root canals, or intervertebral foramina. Two broad groups include acquired (usually caused by degenerative changes) and congenital or developmental. Some prefer the term, clinical stenosis, reflecting the importance of symptoms in establishing the diagnosis. The imaging changes in general are more extensive than expected from the clinical findings. There is no strong relationship between degree of stenosis and patient symptoms. Specific imaging findings do not predict benefit from surgery. Position-dependent (dynamic) stenosis that worsens in extension and improves with flexion can be distinguished from static stenosis.

Congenital stenosis is explained primarily by shortened stubby pedicles with flattened axial appearance of the bony canal resulting from decreased anterior-posterior diameter along with narrowing of the lateral recesses and foramina. Congenital stenosis usually is asymptomatic but reduces the reserve of the spine to accommodate degenerative hypertrophic changes during aging. Acquired central canal stenosis in older patients typically is associated with disk and facet disease, disk bulges, and ligamentous hypertrophy and laxity. Degenerative spondylolisthesis and scoliosis also can contribute significantly to spinal stenosis. CT myelography with axial and multiplanar reformatted images provides greater detail than routine studies. Sagittal and axial MR images with T2 weighting allow accurate assessment of thecal sac compression.

Acquired lateral recess stenosis is caused by disk–end-plate or superior articular facet hypertrophic changes. Asymptomatic nerve root contact may be seen with disk bulgings, protrusions, and osteophytosis. Contact by disk extrusion, however, is a significant finding. Axial MRI or CT myelography shows a triangular-shaped normal lateral recess. Facet and disk margin hypertrophic changes cause acute angled narrowing, whereas isolated facet hypertrophic arthropathy or PEH (which alone can cause radiculopathy) results in trefoil-shaped narrowing.

Acquired foraminal stenosis can be caused by lateral extension of disk bulging when associated with spondylosis, facet joint degenerative hypertrophic changes, or laxity (rostrocaudal subluxation). Loss of disk space height results in decreased vertical dimension of the foramina, whereas the sagittal dimensions are related to the sagittal dimensions of the pedicle and central canal. Therefore, foraminal stenosis commonly is seen in association with canal stenosis. Sagittal MRI provides reliable assessment of the foramina except in scoliosis. CT (preferably CT myelography) can complement the assessment and provide better demonstration of the bony foraminal canals.

Epidural lipomatosis is the accentuation of normal epidural fat, usually seen in the setting of endogenous or exogenous corticosteroid excess, and is common in obese individuals. This usually is an incidental finding, but lipomatosis in the setting of structural stenosis can add to the compromise of the central canal. Rarely, epidural lipomatosis alone can lead to radiculopathy and cauda equina.

Cauda equina syndrome is related to simultaneous compression of multiple lumbosacral nerve roots below the level of the conus medullaris, resulting in a characteristic pattern of neuromuscular and urogenital symptoms. There are many potential causes, including epidural tumors, disk herniations, and epidural hematomas. Disk herniations account for approximately 1% of cases. Acute onset of symptoms is a medical and surgical emergency. MRI and myelography can demonstrate compression of the thecal sac reliably, whereas MRI also can characterize the cause of compression .

- **Addendum**

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